INDEX NUMBER

Vol. 66, No. 12 Established 1884 December 1949

ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

LEADING ARTICLES IN THIS NUMBER

Disorders of Respiration in Bulbar and Other Forms of Poliomyelitis, with Special Reference to Muscle Spasm.
John F. Pohl, M.D. 537

Congenital Heart Diseases.
Hans Moutner, M.D. 546

Napkin Dermatitis Caused by Ammoniacal Urine. Treatment with Methionine.
Louis S. Goldstein, M.D. 553

Anomalies and Diseases of the Umbilical Cord.
Stephen E. Pascucci, M.D. 561

Voice and Voice Problems in Children.
I. W. Karlin, M.D. 567

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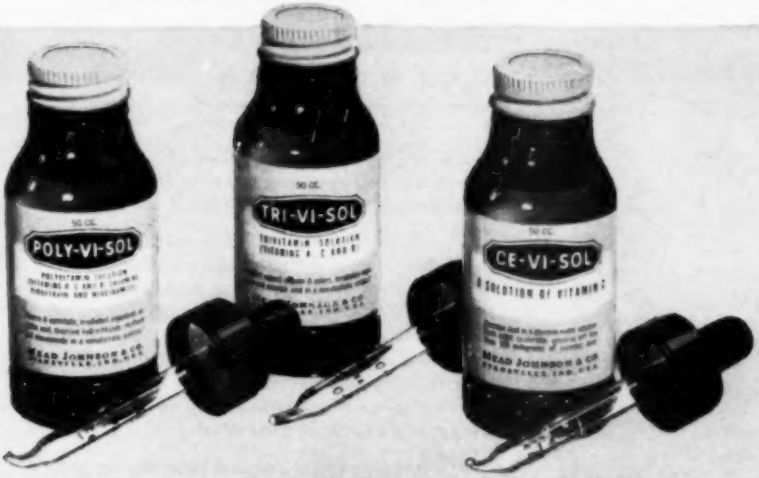

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Business Communications address to E. B. TREAT & Co., INC., 45 East 17th Street, New York 3

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CONTENTS

ORIGINAL COMMUNICATIONS

Disorders of Respiration in Bulbar and Other Forms of Poliomyelitis, with Special Reference to Muscle Spasm.
JOHN F. POHL, M.D. 537

Congenital Heart Disease.
HANS MAUTNER, M.D. 546

Napkin Dermatitis Caused by Ammoniacal Urine. Treatment with Methionine.
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STEPHEN E. PASCUCCI, M.D. 561

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(Continued on page 5)

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(Continued from page 3)

DEPARTMENT OF ABSTRACTS

Nathan, P. W., and Ransford, P. M.: Significance of Convulsions in Infancy. 572

Kaplan, M. Z., and Powelcit, A. C.: Proteus Vulgaris Meningitis..... 572

Shapiro, M. J.: Diagnosis of Congenital Heart Disease by Ordinary Methods. 573

Friedman, E., and Silverman, I.: The Effect of Antihistamine Medication on the Tuberculin Reaction in Children..... 574

Morgan, E. A: Penicillin in the Treatment of Prenatal Syphilis..... 575

Debré, R., and Mozziconacci, P.: Streptomycin Treatment of Septicemia and Meningitis Due to Intestinal Organisms in Infants..... 576

ITEMS

Blalock Operation for Congenital Cyanosis..... 545

Serous Meningitis in Epidemic Pleurodynia..... 552

Milk Embolism as Cause of Sudden Death in Infants..... 560

Penicillin Aerosols in Pediatrics..... 566

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1. Meeuwen, W. McK.: Infant Nutrition, St. Louis, C. V. Mosby Co., 1941, p. 83.
2. Ibid. p. 96.
3. Kugelmass, I. N.: Newer Nutrition in Pediatric Practice, Philadelphia, J. B. Lippincott Co., 1940, p. 625.

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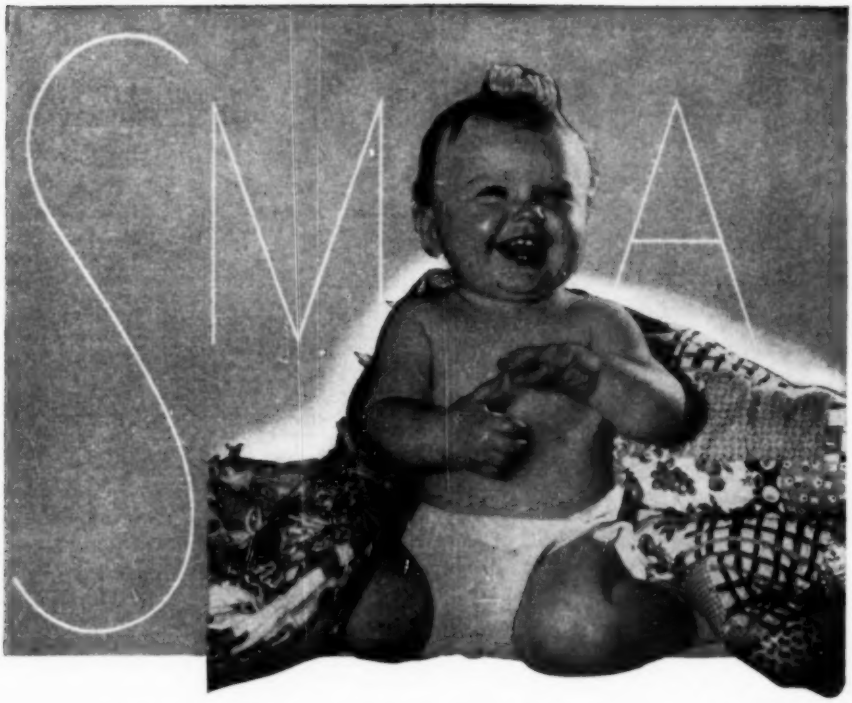

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DISORDERS OF RESPIRATION IN BULBAR AND OTHER FORMS OF POLIOMYELITIS, WITH SPECIAL REFERENCE TO MUSCLE SPASM

JOHN F. POHL, M.D.

Minneapolis.

Paralytic disorders affecting breathing in poliomyelitis are well recognized[1]. Apart from paralysis, muscle spasm is now accepted as a distinctive feature of the neuromuscular symptomatology of the disease. Studies have been reported supporting the belief that spasm is a local disorder of the muscle[2]. The purpose of this report is to present observations on the effect of muscle spasm upon respiration and also to distinguish the various paralytic disorders which affect breathing. During the 1946 Minnesota epidemic an opportunity was afforded to observe 1,125 patients with acute poliomyelitis. Muscle spasm was found in some part of the body in every patient[3]. Paralysis or muscle weakness was present in 736 (65.4 per cent) of the patients but in 389 (34.6 per cent) there was no weakness or paralysis at any stage of the disease. The patients were admitted indiscriminately to two receiving units, but the 368 patients admitted to one unit were chosen for intensive study from the standpoint of breathing disorders and comprise the material upon which this report is based.

There were 43 patients with actual breathing disorders in the 368 patients. This does not include the cases with simple mechanical interference due to retention of secretions in the throat. Four distinctive conditions affecting breathing became evident in study-

ing the patients. These are described under the sectional headings which follow. One or more of the conditions was present in each of the 43 patients. Death accurred in 18 patients, a mortality of 4.9 per cent. Breathing disorders were present in 16 of the fatal cases and absent in 2. In 7 cases death appeared to be due to cerebral involvement (polio-encephalitis) rather than to respiratory failure even though a breathing disorder was present in several of those with fatal encephalitic symptoms.

EFFECT OF MUSCLE SPASM ON BREATHING

Spasm causes muscle tissue to contract and shorten and in this way the condition has an effect upon respiration. Permanent changes in the peripheral tissues resulting from spasm underlie the contractures and deformities of poliomyelitis.[4, 5] The exact nature of muscle spasm has not been established, but published observations of independent investigators support the belief that muscle shortening is intrinsic to the affected muscle tissue itself[2, 6] rather than due to spinal cord disease. Mention should be made in this connection of reports in the literature[7, 8, 9, 10] of primary myocarditis in poliomyelitis. Necrosis and cellular infiltration of heart muscle is described.

In this series of 368 patients, spasm of the chest muscle was found to be either a serious cause of or a contributing factor to breathing difficulty in 38 (88.4 per cent) of the 43 cases with true breathing disorders. It is of importance to emphasize that breathing disorders in poliomyelitis are not confined to patients showing paralysis. Following is a typical case in which the patient at no time showed symptoms of paralysis of any part of the nervous system.

Patient D. M., male, age 7 years, onset of poliomyelitis August 2, 1946. Spinal fluid findings: protein, 23 mgs.%; cells, 20 per cu. mm. When admitted patient complained of severe pain in the chest and difficulty in breathing. Chest expansion was markedly reduced on inspiration. Hot fomentations were intensively applied to the chest for a period of 12 days, when the chest expansion improved and the pain was relieved. The patient was hospitalized for 17 days to relieve all muscle shortening.

Muscle spasm manifests itself by pain, tenderness or muscle shortening. Limitation of excursion of the chest, dyspnea and an

increased rate of breathing are easily mistaken for signs of paralysis. Spasm can be differentiated by palpating the intercostal muscles for tenderness, although this is not infallible. Spasm of other muscles which attach to the ribs, such as the abdominal and spinal muscles, can restrict breathing and must be kept in mind. Spasm and shortening of the pectoral and latissimus dorsi muscles is examined by gently abducting the arm away from the side to determine whether there is pain or any restriction of motion (see Fig. 1). Spasm of the diaphragm causes an exhalatory

Fig. 1. Spasm of pectoral muscle is severely painful and inhibits breathing. Examiner is abducting arm. Note restriction of arm movement due to muscle condition.

type of disorder in which the chest is expanded and the patient's eyes bulge on exhalation in his attempts to rid the lungs of air.

The best treatment of muscle spasm known at present is the application of moist heat. Where the chest muscles are painful and tight, hot foments may be applied every few moments until the patient is relieved. Care must be exercised in applying hot fomentations not to encircle the neck or to apply heavy packs which

would compress the trachea or chest and add to the patient's distress. An important point is to start treatment early, as spasm tends to fibrose muscle tissue and permanently restrict chest movements.

The mechanical respirator with its action of elevating and lowering the ribs aggravates the condition of spasm and at the same time the respirator shell itself makes treatment of the muscles difficult or impossible. Every effort should be made to continue treatment of the chest muscles of patients who have paralysis in addition to spasm and who must be placed in a respirator.

PARALYSIS OF CHEST MUSCLES

Muscular weakness or paralysis of the chest sufficient to impair respiration was present in 12 of the 43 patients in this series with true breathing disorders. The diaphragm is innervated from the fourth cervical level of the spinal cord and the intercostal muscles down to the eleventh thoracic level. In massive infections of the spinal cord of this type there is apt to be paralysis of other parts of the nervous system. Bulbar symptoms were present in 9 (75 per cent) of the patients with paralytic chest muscles. Chest paralysis was the direct cause of death in 4 patients and, in an additional fatality, chest paralysis was an important contributing factor in death. In 83.3 per cent of the 12 paralytic cases having breathing disorders muscle spasm was a factor contributing to the respiratory distress.

The patient with paralysis is observed to have progressive loss of chest excursion with accompanying dyspnea and cyanosis. Respirations are regular but rapid and shallow. A convenient method of determining progress is to ask the patient to take a deep breath and count. Normally the patient will count to 20 or 25 with one breath. When the patient cannot secure enough air to count beyond 10 or 12 there is danger of insufficient oxygenation. Transitory apathy and stupor, which is relieved by administration of oxygen or artificial respiration, differentiates lack of oxygen from infection of the brain (polio-encephalitis).

The mechanical respirator has come to be the accepted treatment for patients with chest paralysis. It is a function of this report to point out that spasm of the chest muscles frequently accompanies paralysis of the chest muscles and that prompt application of

moist heat will often appreciably lessen the patient's difficulties. If the patient still is not sufficiently oxygenated by his own efforts plus the administration of oxygen by face mask or nasal tube there is no recourse other than to use the mechanical respirator.

The respirator was used for 7 patients in this group, with survival in 4 of the cases so treated. The 4 surviving patients were in the respirator only intermittently during the time the respirator was used. In one case the respirator was used for 8 days, and in the others for 17 days, 23 days and for 2 months. The history of of one of the surviving patients will illustrate the method employed in handling respirator cases to preserve the greatest possible function of the respiratory structure.

Patient M. S., age 18 years, onset of poliomyelitis September 1, 1946. On the seventh day of the disease the chest was painful and immobile due to spasm of the pectoral muscles, and the patient was having difficulty getting her breath. The only bulbar symptom was slight weakness of the left side of the face. Intensive hot packing to the chest was applied. On the tenth day the condition was unimproved and was not relieved by oxygen. Weakness of both legs, the shoulders, the spine and of the abdominal muscles had developed and it was suspected that paralysis of the chest was causing part of the respiratory difficulty. The patient was placed in the respirator to improve oxygenation. It was possible to open the respirator at hourly intervals throughout the day and apply hot foments to the chest. By the tenth day in the respirator the patient was able to stay out for two half hour periods daily while attempts were made to release the tight pectoral muscles and mobilize the thorax. The respirator was discontinued after being used for 23 days. Chest excursions eventually became good although some shortening of the pectoral muscles persisted. The spine remained rigid due to the impossibility of applying treatment for spasm to this area while the patient was in the respirator. After 18 months hospitalization, one leg remained completely paralyzed, the shoulders remained weak and the abdominal and spinal muscles showed severe residual weakness. Joint contractures had been prevented and the patient was able to walk fairly well without assistance.

Patients who must be treated in respirators for long periods of time usually tend to become severely crippled from contractures. The chest muscles also were observed to be tender and sensitive for weeks or months in patients who have been in the respirator. Our experience with respirator cases has been disappointing in

that these patients succumb readily from minor causes and seem unable to withstand complicating infections.

BULBAR POLIOMYELITIS

Paralysis of the respiratory center in the medulla proved to be a highly fatal condition. All of the seven patients in this series with such involvement died. It is commonly stated that respirations in the bulbar patient tend to become irregular in rate and rhythm. This did not appear true in our cases. Respirations were regular as long as the patient was able to breathe, and no difficulty with synchronization was encountered in the two bulbar patients who had to be placed in the respirator because of paralysis of the chest muscles. The cyanosis which appeared in these patients was associated with falling blood pressure and weak rapid pulse suggestive of circulatory failure. The respirator did not relieve cyanosis and did not restore spontaneous respirations in either of the two patients in which it had to be used.

The most common form of bulbar poliomyelitis paralyzes the cranial nerve nuclei and causes ocular muscle disorders, facial weakness, nasality of voice, pooling of secretions in the throat, regurgitation of fluid through the nose and difficulty in swallowing. These symptoms frequently precede respiratory center involvement and are warning signals. In this group of 368 patients, cranial nerve symptoms appeared in 73 (19.8 per cent) of the cases.

The upper cranial nerves down to and including the eighth nerve innervate the ocular, facial and masticatory muscles, and paralysis of these nerves does not directly affect breathing. Those of the lower group, below the eighth nerve, concern voice and swallowing, and paralysis of these nerves creates problems in the management of respiration. Of the 73 patients with bulbar cranial nerve involvement the upper group were affected in 13.7 per cent and the lower group in 54.8 per cent, while in 31.5 per cent there was a combination of involvment of both the upper and lower groups. In 20 patients the total paralytic involvement was limited to the cranial nerves and none of these patients died, but in the remaining 53 patients other parts of the nervous system became paralyzed in addition to cranial nerve affection.

One of the problems in the bulbar patient is to keep the throat clear of accumulated secretions which cannot be swallowed and

which interfere with breathing. This is satisfactorily accomplished by frequent suctioning with a soft rubber catheter. It had been difficult to account for the excessive amount of secretion which collects in the throat until in one case it was discovered that secretions were welling up from the stomach. It is now believed that much of the secretion comes from the esophagus. In several patients it was demonstrated experimentally that water introduced by catheter into the esophagus would not flow into the stomach but could easily be retrieved from the esophagus with a syringe and catheter. In the presence of paralysis of the esophagus, food and fluid should be kept out of the patient's mouth so as to avoid aspiration into the lungs. Prone postural drainage, lowering the patient's head over the edge of the bed, proved helpful in clearing the throat and esophagus. As soon as the patient demonstrated ability to swallow and retain small amounts of fluid, the passage of secretion into the stomach was encouraged by elevating the head or sitting the patient up.

Spasm of the muscles of the neck and chest was found to be a complication in many bulbar cases. Chest muscle spasm was associated with bulbar disease in 21 (28.8 per cent) of the 73 bulbar cases. Occasionally, spasm of the posterior neck muscles was of such severity as to cause head retraction and this interferes with swallowing. Spasm of the jaw muscles affects the ability to open and close the mouth, thus interfering with swallowing. The following case report is illustrative:

Patient C. S., male, age 10 years, onset of poliomyelitis July 28, 1946. Spinal fluid findings: protein 73 mgs. per cent; cells 44 per cu. mm. At the height of the disease on the fourth day, there was slight convergent strabismus of the eyes, mild nasality of voice and slight facial weakness, symptoms which were due to palsy of the cranial nerves. No muscle weakness developed otherwise. The jaws were tightly adducted and could be pried apart only one-half inch. When the patient attempted to open his mouth the mandible moved to the right and this condition was interpreted as due to spasm of the masseter muscles, particularly the right. Hot packs applied to the face considerably relieved the spasm of the jaws within 10 days, but slight spasm persisted for three months. When the patient was last examined on April 20, 1948, 21 months after the onset, slight facial weakness was still present but the jaw muscles were normal.

The paralyzing effect of poliomyelitis in bulbar cases was found

frequently not confined to the bulbar area but also involved the spinal cord. In 9 of the bulbar cases the cervical or thoracic spinal cord was diseased with resulting weakness or paralysis of the chest muscles. These patients fatigued readily and in one instance it was necessary to use the respirator. This was accomplished satisfactorily.

POLIO-ENCEPHALITIS

Involvement of the cerebrum proper was regarded as one manifestation of poliomyelitis rather than as a separate disease. Of the 368 patients in this study 26 showed signs of encephalitis, and this was believed to account for death in 7 (38.9 per cent) of the 18 fatal cases.

Respiration appeared to be affected in a characteristic manner. Early the patients were observed to be fretful and irritable but without breathing changes. Later there was a tendency to lapse into listlessness and apathy. At this stage respirations became irregular in rate, depth and rhythm, and resembled Cheynes-Stokes breathing. There was no cyanosis at this stage. A few patients expired in this listless state. In most cases, with further invasion of the sensorium, the patients became anxious and excited and developed a fear of impending death. This was followed by delirium, confusion, restlessness and unconsciousness. In some cases the respirations then became regular but stertorous. At this stage the patients developed a mottled cyanosis that was not relieved by the administration of oxygen.

Of the 26 patients with encephalitic symptoms, there was involvement of other parts of the nervous system in 96.1 per cent, as evidenced by muscle weakness or paralysis. In two cases, with apparent bulbar and high spinal cord involvement, an attempt was made to place the patient in the respirator. This was unsuccessful as the patient would not cooperate and the respirator could not be synchronized with the patient's own breathing. As a result of the experience with this and other groups of patients of this type it is believed that the respirator is contraindicated in cases where encephalitic symptoms supervene or become predominant.

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1945 *Medical Arts Building.*

BLALOCK OPERATION FOR CONGENITAL CYANOSIS. (Lyon Chirurgical, 44: 129, March-April 1949). Santy and co-workers performed the Blalock operation on 23 boys and 22 girls, between the ages of 23 months and 15 years, from a group of 100 children with congenital cyanosis. The operation was performed only in those children in whom deficient pulmonary circulation had been diagnosed on clinical and roentgenologic examination. In one case in which the Blalock operation was attempted, it could not be performed because no pulmonary artery was present on the surgically treated side. In all patients operated on the subclavian artery was used for the anastomosis with the pulmonary artery. As a general rule the anastomoses were carried out by the end to side technic. The anastomosis of the subclavian artery with the pulmonary artery by the end to end method of suture warrants satisfactory functional results and is to be considered safe in difficult cases in which the end to side method appears to be technically impossible. There were 5 operative deaths, 3 from cardiac syncope, and 2 from diffuse venous hemorrhage. Five additional patients died in one to thirteen days after the operation, 2 of them from pulmonary edema, 1 from septicemia and 1 from empyema. In 3 operation failed, in 1 because of absence of the right pulmonary artery and in 2 because of the poor permeability of the anastomosis. The operation was successful in 32 patients with complete transformation of the clinical and roentgenologic symptoms. —*Journal A.M.A.*

CONGENITAL HEART DISEASES*

HANS MAUTNER, M.D.
Wrentham, Mass.

The history of the congenital heart diseases is readily divided into three periods. The first was devoted almost wholly to anatomical studies, the second to the genesis, which includes embryology and physiology; the last to therapy. This article will consider the first and second periods only.

The first period probably began with Galen to whom the existence of the foramen ovale and congenital cyanosis was not unknown. Stenonis (1671) described a condition now called the tetralogy of Fallot. Sandifort, in 1777, described a similar type of heart. The first case of an isolated defect in the ventricular septum was described by Pozzi in 1673. Two hundred years ago interest in cyanosis started with Senàc who explained this state as due to a mixture of arterial and venous blood. Morgagni, on the other hand, stressed that cyanosis was produced by stagnation of blood in the lungs. At first Senàc's explanation was widely accepted almost to the exclusion of Morgagni's ideas. But in 1826 Louis confirmed Morgagni's interpretation and renewed its importance. Peacock, in 1854, first demonstrated that cyanosis in many children develops at a later stage; that the blueness appears only after the onset or aggravation of the heart failure and is due to the faulty pulmonary circulation. Later it was established that persistent cyanosis is caused by dilatation and enlargement of the peripheral capillaries. This was observed on the ocular fundi by Holloway (1912) and in the fingers by Jansen (1920).

Many methods and instrumental devices have been employed for diagnosis. Of all methods, the value of percussion and auscultation can not be over-estimated. Taussig, in her textbook, depends chiefly on x-ray and fluoroscopy examination. Castellanos introduced contrast filling of the large vessels with diodrast. Catheterization of the heart chambers and the large vessels was introduced by Forssmann and O. Klein and applied in the diagnosis of congenital heart diseases by Cournant, Dexter and others. By this procedure the pressure and oxygen saturation in different parts

*Paper read before the Medical Circle, New York Academy of Medicine, May 1949.

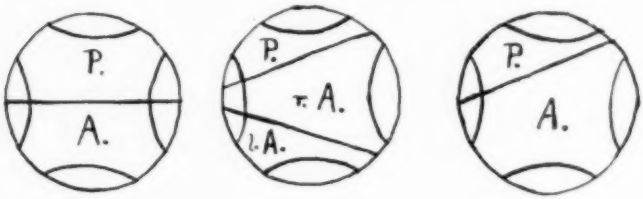
of the heart are determined and this information contributes to an exact diagnosis. Roentgen-kymography, as improved by the method of fluorocardiography, introduced by Henny, Boone and Chamberlain, later modified by Luisada and Fleischner and others, is another useful method.

The second period contained outstanding contributions by Meyer, Rokitansky and Spitzer. The leading pediatrician of his time, H. Meyer, in 1857, wrote a treatise on the transposition of the large vessels and concluded that merely a defect in the rotation of the vessels themselves accounted for these congenital malformations. Rokitansky (1875), on the other hand, stressed that it was chiefly a defect of rotation of the septum in the arterial truncus. For the first time the emphasis was placed on the embryology of the heart and the large vessels. This theory was welcomed with great enthusiasm but very soon objections appeared. Born, His and others showed that the last part of the ventricular septum which comes to be closed is not supplied by the ventricular but by the truncus septum.

I had the opportunity, in 1920, to observe a boy in the Vienna Karolinenkinderspital who had a malformation of the heart with dextrocardia and intense cyanosis and a concomitant tracheomalacia. The autopsy revealed a cor triloculare with some septal remnants in the one large ventricle. The aorta and the pulmonic artery came from the right side. At that time Dr. Spitzer had devoted years on the embryology of the heart and I turned to him for an explanation of the anomaly that confronted us. His explanation of the specimen, published by Mautner and Loewy and enclosed together with photograph by Abbott in her atlas of congenital heart diseases, was that the right ventricular aorta has persisted. Some weeks later I had occasion to visit Dr. Spitzer with a more common case of malformation, a typical tetralogy of Fallot. The heart had been partly opened. Spitzer examined it carefully and predicted that it would have to have two pulmonary semilunar valves. This proved to be the case.

Spitzer began to investigate why the mammalian heart is constructed in such a complicated way. He compared it with the more simple heart of fishes and believed that respiration is responsible for the need of a double pump. Optimal oxidation is obtained when all the blood flows through the lungs. A simple division of

the heart by a septum would, therefore, not be sufficient without a switch from right to left. This switch is accomplished by a torsion of the heart itself around its axis for 180°. The torsion occurs at an early stage of development when the division of the truncus is accomplished but the pulmonary veins are side-branches of the vena cava and reach the heart in a later stage of development. Spitzer demonstrated this normal torsion of the heart and the relation of the three proximal bulbus cushions A, B, and C to the different parts of the septa. Development of the cushion C is important in reptiles as long as the heart has two aortae, one from the left and one from the right ventricle; the ventricular septum is not completed in reptiles. In the right ventricle two muscle ridges are visible which resemble a septum in systole only. The normal torsion of the mammalian heart is probably the reason for the occlusion of the "other" aorta of the right ventricle. The



Mammals Reptiles Tetralogy

two muscle ridges now become superfluous and are reduced to two small ridges, the crista supraventricularis and the trabecula septomarginalis. An incomplete torsion of the heart in an early stage of development prevents the occlusion of the aorta of the right ventricle, at least in its intracardial part, and the muscle ridges remain larger. In the tetralogy of Fallot, a common heart malformation, the intracardial out-flow of the right ventricular aorta remains open and combines with the aorta of the left ventricle and thus prevents the occlusion of the septum; the small pulmonary artery is a direct consequence of it. The truncus always contains four distal cushions, from which the truncus septum and the semilunar valves originate. In mammals two opposite cushions build up the septum and in both large vessels the remaining parts of the cushions build three valves. In reptiles three vessels are

present. Therefore, two septa are necessary, leaving over material for two valves in all three vessels. The two aortae are combined in tetralogy of Fallot, using up two-thirds of the truncus and leaving one-third only for a small pulmonary artery, resulting in three valves in the combined aorta and two in the pulmonary artery.

The different types of malformation of the heart depend on the degree of torsion established and how much has been omitted. For the latter Spitzer uses the term "detorsion." When the torsion has not reached the stage associated with tetralogy, the ventricular septum becomes even smaller and the muscle ridges in the right ventricle increase. It would appear that these ridges are the septum and that the pulmonary artery arises from the left of it. This combined with an opened right ventricular aorta gives the impression of a transposition of both vessels. Spitzer has stressed that the many variations of heart malformations make it necessary to assume that partial situs inversus of the atria and of the aorta may also occur. Normally, the aorta develops from the fourth branchial artery, and the left subclavian from the fourth left branch. Persistence of the right aorta is not too rare in man. In the case of tetralogy the right ventricular aorta becomes closed somewhat more distally than in the norm. In more complicated cases more of this aorta may be present or even the entire abnormal aorta is developed. These are cases reported with both aortic arches persistent, causing breathing difficulties, etc. Sometimes one aorta is present, but the subclavian artery originates at an abnormal point, and may cause similar trouble. Gross has treated these by the simple expedient of tying off one of the vessels.

Spitzer's four types of transposition do not cover all the possibilities and nearly every case differs in some details. This appears reasonable since torsion has so many variations. The cardinal point in Spitzer's theory is that transposition occurs by an incomplete torsion of the heart itself and not by irregularities in the torsion of the vessels or the septa of the vessels. This torsion of the heart accounts for the twisting of the large vessels one around the other.

Incomplete torsion of the heart explains transpositions, the tetralogy and isolated defects of the ventricular septum. Other malformations do not depend on this "detorsion." Even some of the ventricular septal defects must develop on another mechanism.

The incomplete torsion opens the upper part of the septum, in the region of the pars membranacea. It does not explain defects at a lower portion of the septum, the so-called "piercing" defects. This descriptive term was chosen because it was assumed that these defects are found when the high left intraventricular pressure breaks through the septum, a very improbable event. Moenckeberg speaks about intratrabecular defects. The septum is composed of some parts which normally fit together but sometimes leave small apertures which become closed by muscle action. Moenckeberg found such defects relatively often in young children but never in adults, and supposed that they closed spontaneously by endocardial growth. These defects are probably of the type which produce a very loud congenital murmur, which surprisingly enough may disappear completely. The following unusual case illustrates this point.

One of female twins showed at birth a large heart and a very loud systolic murmur heard over the lower part of the sternum. At approximately two months of age the heart, while under a fluoroscopic examination, suddenly shrank in size and the murmur disappeared and never returned. The severe crying of the infant may have played a part in muscular closure of the defect.

Defects of the atrial septum are probably caused by other mechanisms.

The ductus arteriosus remains patent when its usual closure immediately after birth is prevented. This is, therefore, strictly speaking, not a congenital state. Strassmann has shown that the duct springs from the aorta at an angle and that when respiration starts and the quantity of blood in the pulmonary artery increases, the angle between duct and aorta becomes smaller, the wall of the aorta acting as a valve to close the duct. Haberda believed that the muscles of the duct contract and thus close the duct directly. Others have attributed the closure to a coagulation of the blood in the duct following birth. It seems probable that all three mechanisms are effective. Any disturbance of these mechanisms may produce a permanently patent duct.

The mechanism leading to coarctation of the adult type is well understood. Bonet described a newborn as well as an adult type. The duct is open in the newborn type and closed in the adult type.

The newborn type is usually associated with other severe cardiac malformations, whereas the adult type is probably associated with occlusion of the duct. The muscles of the duct normally disappear, the duct being reduced to connective tissue only. Sometimes this change extends into the aorta at the site of origin of the duct, raising, however, no obstruction to blood flow. Since this connective tissue ring does not keep in step with the growth of the aorta, stenosis ensues. The stenosis becomes more marked at puberty when heart and aorta receive a special impetus in growth. This is the reason why this type of coarctation does not exist in the newborn.

Some malformations seem to be the result of fetal endocarditis. German measles in the pregnant mother is now recognized as a cause of severe cardiac malformation. Other virus infections, especially measles, may act in a similar way. Hagströmer has recently claimed that measles may cause malformation in the fetus of an immune mother exposed to measles. The two cases of malformation of the heart following German measles of the pregnant mother which I have seen at autopsy were of a type which suggested an endocarditis and had small resemblance to the malformations of Spitzer's series. One showed a stenosis of the pulmonary valve with a septal defect. It differed from the tetralogy in that it showed a marked valvular change and the pulmonary artery was normal in size. It seems likely that endocarditis affected the valve in a stage of development of the heart when the septum was not completed and the stenosis prevented the normal occlusion of the defective septum. Many years ago I saw a child born to a mother who had acute endocarditis. The newborn showed the typical excrescences at the mitral valve observed in adults with this disease. Isolated occlusion or severe stenosis of the valves without other complications may be the sequel of fetal endocarditis at a later stage when the septa are completed.

Lutembacher's syndrome, the congenital stenosis of the mitral valve combined with a defect of the atrial septum has also been interpreted as the sequel of endocarditis, with mechanical prevention of the occlusion of the atrial septum. However, Dressler and Roesler doubt this interpretation and consider the defect of the atrial septum the primary abnormality.

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SEROUS MENINGITIS IN EPIDEMIC PLEURODYNIA. (Schweizerische medizinische Wochenschrift, Basel, 79, 241, March 19, 1949). Gsell reports 7 cases of serous meningitis complicating epidemic pleurodynia in 5 patients between the ages of 16 and 40 and in 2 children aged 5 and 7. These cases occurred in the Swiss canton of Saint Gallen during the summer and autumn of 1948. The term myalgic meningitis has been suggested for this type of virus meningitis. Serous meningitis may occur in single cases of familial epidemic pleurodynia as in 1 of the author's cases in a family of 10 with pleurodynia, or it may occur sporadically without additional incidence in the same household as in 4 of the author's cases. In all 7 patients the meningitis occurred during the second bout of the disease between the third and sixth day. The clinical picture corresponded with that of a more or less intensive meningitis without pronounced psychic changes. There were no encephalitic complications; the maximum cerebrospinal fluid cell count varied from 17 to 408. Neutrophils were predominant in the cerebrospinal fluid in the early stage and lymphocytes in a later stage. There was a slight increase in the albumin content; the sugar content was normal. The blood picture showed a tendency to leukopenia. The blood sedimentation rate was only slightly increased. The duration of the meningitis varied from one to three weeks. All patients recovered without any sequelae. Treatment consisted of rest in bed, lumbar puncture and administration of analgesics. Chemotherapy and antibiotics are of no value. The increased incidence of epidemic pleurodynia in Central Europe during the last year is emphasized.—*Journal A.M.A.*

NAPKIN DERMATITIS CAUSED BY AMMONIACAL URINE. TREATMENT WITH METHIONINE

LOUIS S. GOLDSTEIN, M. D.
Yonkers, N. Y.

This paper is an extension of previously reported studies[1] in which methionine was employed for the treatment of napkin dermatitis (known also as diaper rash and ammonia dermatitis) affecting infants and young children.

The clinical picture of napkin dermatitis is well-known and has been frequently described in medical literature.[2, 3] According to Rhame,[2] the diagnosis is simple, the odor of ammonia always being present in a greater or lesser degree, depending on the concentration in the urine. The lesions consist of inflamed areas in the region in contact with the diaper (inner thighs, genitalia, buttocks and lower abdomen) and are usually characterized by blebs and vesicles. If permitted to go unchecked they may develop into severe ulcerations, which are particularly troublesome in the external urinary meatus. Lesions may also develop on the infant's calves and heels.

In many cases the disorder is also characterized by frequent cyclical recurrences, and the excretion of a highly colored acid urine which stains the diaper.[1]

ETIOLOGY OF AMMONIACAL URINE

According to Cooke,[4] ammonia is produced by the bacterial decomposition of urinary urea. The responsible organism is B ammoniagenes which occurs in the feces and finds a good culture medium for growth in the diaper wet with urine.

This mechanism has received wide acceptance among physicians as being responsible for the formation of urinary ammonia. Clinical literature reveals, however, that there are other etiological factors which play significant roles in ammonia dermatitis, and toward which therapy should be directed. One factor appears to be the metabolic "balance disturbance" which results when the infant is fed excessive amounts of fat.[2, 3, 5, 6] In such cases the ammonia is formed internally as a result of the metabolic disturbance and is often associated with constipation, irritability and other signs of gastro-intestinal disorder. Allergic disturbances

The author is Director of Pediatrics of the Professional Hospital in Yonkers.

have been described as being another etiological factor of perhaps even greater importance.[1] It has been observed that ammoniacal urine occurs more frequently in artificially fed infants than it does among breast fed infants.[2, 3, 4]

A later section of this paper will contain more detailed discussion of the mechanisms through which excess metabolic ammonia may be produced and excreted in the infant's urine.

TREATMENT

In an earlier study[1] it was noted that oral administration of thiamine (60 mg. daily in the infant's formula) was effective in clearing the urine of ammonia and in bringing about improvement in the dermatitis. Inasmuch as this effect could not be explained in terms of thiamine's known vitamin activity, other substances (without vitamin activity) were tried in an effort to elucidate the mechanism of this action. Methionine was selected for trial because its structural configuration bears certain resemblances to that of thiamine; both contain sulfur, methyl and amino groups. Results were equivalent to those obtained with thiamine.

A series of trials was made with choline and creatine, both of which contain amino and methyl groups, but no sulfur. No response was obtained with these compounds, suggesting that the therapeutic activity of the thiamine and methionine is due to the presence of the sulfur contained in these molecules.

In subsequent studies, methionine rather than thiamine was used because it is easily administered, has a more acceptable taste and odor, and produces less gaseous discomfort.

A satisfactory response was obtained in a majority of cases in which methionine was employed, using a dosage of 3 grains daily, given in the infant's formula. If the dermatitis did not show improvement within one week after therapy with methionine was instituted, the dosage was increased to 6 grains daily. A group of 50 infants were given daily prophylactic doses of 3 grains of methionine starting at the age of two months and continuing for six months. At no time did these infants exhibit ammoniacal urine or napkin dermatitis.

CASE STUDIES

Since the publication of the first report,[1] methionine has been

employed for the treatment of over 500 infants exhibiting ammoniacal urine. Methionine, granulated* (10 per cent dl-methionine plus 90 per cent dextrose and lactose) was used in all cases. One teaspoonful of methionine, granulated, contains 3 grains of methionine, which represents the daily dose for the infant under one year of age. For infants one to two years old, 1½ to 2 teaspoonfuls of methionine, granulated (containing 4½ to 6 grains of methionine), are required daily. This preparation is soluble, palatable and stable under adverse weather conditions; it mixes readily in the daily milk formula.

Of the more than 500 cases treated with methionine, granulated, therapy was successful in all but three. The ammonia consistently disappeared from the urine in three to four days; napkin dermatitis and ulcerations (including those of the external urinary meatus) cleared up in seven to ten days. Therapy was continued for at least one week. With the appearance of cyclic recurrences, methionine, granulated, was prescribed for another week or two. Application of zinc ointment was the only local therapy employed.

The three cases of failure were noted as follows: one infant refused the formula because of flavor; a second vomited the formula; the third developed a generalized macular dermatitis indicative of a sensitization reaction similar to that observed following the administration of sulfonamides. Sensitization to amino acids is generally considered highly unlikely; this case represents an instance of such sensitization. In these three cases, 60 grains of thiamine were substituted for the 3 grains of methionine, and excellent results were obtained.

FIVE ILLUSTRATIVE CASE REPORTS

Case 1. D. H. was a male infant who had an uneventful neonatal hospital stay. On the tenth day, however, a very fine, generalized maculopapular dermatitis was observed. With the onset of the rash, the infant became irritable, developed frequent and prolonged attacks of hiccough, strained for long periods at stool, and cried strenuously from six to nine o'clock every evening. His formula, which consisted of evaporated milk, was changed to

*The methionine, granulated, used was Metiogran, a product of The Debruille Chemical Corporation, 1841 Broadway, New York 23, N. Y.

double-boiled whole milk (the milk is heated for 2 hours and the coagulated protein is skimmed off the top), with subsequent amelioration of symptoms. At two months of age, many discrete papules and papulo-vesicles appeared on the cheeks (mild infantile eczema). Growth and development continued normally. At eleven months of age, he secreted dark yellow, ammoniacal urine which stained the diapers and produced a diffuse erythema of the napkin region. The pungent odor of ammonia pervaded the atmosphere of his crib and room. These symptoms persisted until he was one year of age. At this time one teasponful of methionine, granulated, was added to his daily milk ration. Within three days, the ammonia disappeared. The dark yellow color of the urine persisted.

Case 2. E. D. was first seen at one month of age. He exhibited a generalized erythematous macular dermatitis plus numerous impetiginous lesions on the torso. There was considerable oily scaling of the scalp. He cried frequently, had frequent hiccough, and strained so forcibly at stool that his face became quite red. At eight months of age, postauricular intertrigo (Kugelmass' sign) became apparent. When he was one year old, he secreted dark yellow urine which was strongly ammoniacal. One teaspoonful of methionine, granulated, was added to his total daily milk ration. Within two days, the ammonia completely disappeared. The dark yellow color of the urine continued.

Case 3. J. F. was said to be a very hungry infant. She cried frequently and demanded feedings every hour or two. She would finish her bottle and fall asleep, only to awaken one hour later, crying, drawing up her legs, and mouthing as though hungry. There was no history of a primary milk rash on the tenth day. Hiccough appeared frequently and she strained strenuously at stool. At two months of age, multiple lichenoid papules were observed on the cheeks, as well as a fine macular dermatitis on the neck. At seven months of age, she had occasional ammoniacal urine and moderate napkin dermatitis. The following month, the urine became dark yellow and was strongly ammoniacal. One teaspoonful of methionine, granulated, was added to her daily milk ration. Within two days, the ammoniacal odor completely disappeared. The dark yellow color of the urine persisted.

Case 4. C. G. was a one-month-old male infant who was con-

stipated, cried all day long, exhibited frequent hiccough, strained at stool, and cried when he voided. At two months of age he vomited frequently. On the cheeks were many papulo-vesicles. At one year of age the urine became darkly colored and smelled strongly of ammonia. On two occasions he passed bright red blood in the urine. There was neither napkin dermatitis nor obvious ulceration of the external urinary meatus. In two days following the institution of therapy with methionine, granulated, the urine became ammonia-free. There was no further recurrence of hematuria. Therapy with methionine, granulated, was continued for one month.

Case 5. D. W. was seen for the first time at nine months of age. Multiple erythematous papules were present on the abdomen and scrotum. There was obvious ulceration of the external urinary meatus. The mother described a generalized dermatitis which had appeared on leaving the hospital on the tenth day, but there was no further history of eczema during infancy. At eight months of age, the urine became strongly ammoniacal and there was ulceration of the external urinary meatus. One teaspoonful of methionine, granulated, was added to the daily milk ration. Within two days, the urine became ammonia-free; in one week the ulcer of the external urinary meatus was healed.

DISCUSSION

The kidneys have been recognized as the most probable site of ammonia formation.[7] The chief source of the excess observed in the urine appears to be glutamine which breaks down to produce ammonia as a defense against acid intoxication.[8, 9]

Considering, then, the view that ammoniacal urine is produced by excessive intake of fat, it would seem that acidosis and ketonuria occur when the infant cannot completely oxidize the fatty acids released during fat metabolism. There is a loss of alkali from the blood and body tissues, and ammonia is formed to neutralize the excess acid. Thus, it has been observed that ammonia excretion can be lessened or eliminated by reducing the fat intake and by the administration of carbohydrate, protein or alkali.[5, 6]

The effect of methionine as an inhibitor of ammonia excretion is probably due, at least in part, to its glycogenic function. Butts and his co-workers[10, 11] have shown that certain amino acids are

glycogenic and therefore effective in combating experimental ketonuria. It seems reasonable to assume that the ingestion of methionine will result in the production of carbohydrate, thus sparing other metabolites, and reducing the likelihood of acidosis.

Over and above all of the foregoing (though undoubtedly related to it), another etiology can be considered for ammoniacal urine, namely, that it is a manifestation of certain allergic disturbances in the infant.

Glaser[12] has recently pointed out that the introduction of a foreign protein (as contained in cow's milk or in any special formula) into the diet of the infant may be likened to the injection of a foreign protein into an experimental animal. For both there will be an "incubation" period during which antibody formation takes place, and finally there will occur, anaphylaxis in the animal, symptoms of sensitization in the child.

Starting at the day of birth, the infant is constantly being called upon to make adjustment to changes in diet and environment. Sensitization reactions may occur as a result of overindulgence in certain foods on the part of the mother during pregnancy, new foods or drugs passed to the infant through mother's milk, changes in formula, and a change in environment, such as occurs when the infant leaves the hospital. Each change of this kind may be considered a critical period in the life of the infant, and these periods are often associated with dermatological, gastro-intestinal or upper respiratory disturbances, allergic in nature.

Cohen and Rudolph[13] have pointed out that respiratory disturbances caused by allergy in young children are characterized by periodic recurrences. Glaser[12] notes that urticaria often occurs during the first few days of the infant's life, and that colic may develop as an allergic reaction during the first few weeks and reappear periodically until the infant is about three months old. As the child grows older it begins to exhibit the symptoms of respiratory allergy.

During any of these "critical periods" the infant may suffer from sneezing, nose block, coughing, straining at stool, hiccough, dermatitis and digestive disturbances. Such periods may be further characterized by ammoniacal urine and napkin dermatitis. In the majority of cases, on which this and the preliminary report[1]

are based, ammonia dermatitis appeared concomitantly with the allergic manifestations described.

In view of the foregoing, a complete explanation of the manner in which methionine reduces and eliminates ammonia excretion probably cannot be given at present. Anaphylaxis has been shown experimentally to produce structural and functional changes in the liver.[14, 15, 16, 17] Damage to the liver may impair the ability of that organ to synthesize urea from ammonia, leading to an increase in ammonia excretion. Methionine is recognized as being valuable in the treatment of liver damage and may help to restore normal function in this respect.

It seems not unlikely, also, that an allergic flare-up of the type described is associated with disturbances in protein metabolism leading in turn to an acidotic condition. In this circumstance, methionine would be effective, again, by virtue of its glycogenic activity.

Note: It has been stated that sulfur appears to be required in the molecule, for therapeutic results to be obtained. In this connection, it is of some interest that Borsook and his associates,[18] studying the metabolism of thiamine containing radioactive sulfur, found that the sulfur reappeared in the urine in neutral sulfur compounds and inorganic sulfates.

A statement by Rosenberg[19] is also of interest: "There is the possibility that organic chemical compounds containing sulfur belong to the class of protective foods and should be classified as vitagens. Sulfur-containing organic compounds act not only as structural building units, for example, in hair and nails, but are also of specific and functional importance for growth and maintenance of life."

SUMMARY AND CONCLUSIONS

There are multiple causes for ammoniacal urine and napkin dermatitis in infants. Allergy appears to be an important etiological factor. Methionine and thiamine effectively inhibit the formation of excess metabolic ammonia and its appearance in the urine.

More than 500 infants with napkin dermatitis have been treated with methionine, granulated (10 per cent dl-methionine plus 90 per cent dextrose and lactose). Therapy was successful in all

but three cases. The excess ammonia consistently disappeared from the urine in three to four days. Napkin dermatitis and ulcerations cleared up in seven to ten days. In the three cases which could not be treated with methionine, thiamine was substituted and good therapeutic results were obtained.

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189 *Valentine Lane.*

MILK EMBOLISM AS CAUSE OF SUDDEN DEATH IN INFANTS. (Annales Paediatrici, Basel, 171:313, Nov.-Dec. 1948). The cause of death is frequently obscure in sudden deaths in infants. Bronchopneumonia, pneumonia and enlarged thymus are frequently mentioned as causes. Marie says that clinical and anatomic observations have convinced him that there is a simple mechanism not considered by earlier investigators, a milk or food embolism in the pulmonary airways. Such an embolism is produced by the aspiration of the gastric contents into the lung, which may occur in the course of vomiting or of regurgitation that may not even be expelled externally. It is especially likely during vomiting in weak infants or in those with infections. The author describes the histopathologic changes caused by the milk or by the gastric contents in the bronchi and the alveoli and suggests that even if the definite cause of death has not been suspected, the proof may be furnished by careful microscopic study of the lungs.—*Journal A.M.A.*

ANOMALIES AND DISEASES OF THE UMBILICAL CORD

STEPHEN E. PASCUCCI, M.D.
Abington, Pa.

The umbilical cord connects the fetus with the placenta and conveys fetal blood to and from the latter. It is formed from the abdominal pedicle of the embryo. It contains two umbilical arteries, the umbilical vein, the rudimentary allantois, the remnant of the omphalomesenteric duct and a variable amount of Wharton's jelly. The blood vessels are functionally closed, but are still patent anatomically for a period of twenty to twenty-five days. The umbilical arteries become the lateral umbilical ligaments; the umbilical vein, the ligament teres; and the ductus venosus, the ligamentum venosus. During this interval the umbilical vessels are potential portals of entry for infection. The amount of Wharton's jelly determines the amount of granulation tissue which remains when the umbilical cord sloughs.

ANOMALIES

1. *Congenital Omphalocele.* This malformation has been designated by many names, such as "hernia into the umbilical cord," "umbilical eventration," "amniotic hernia" or "exomphalus."

From the sixth to the tenth week of the fetal life the celomic cavity has a forward expansion into the base of the umbilical cord which contains loops of intestines. After the tenth week the abdomen enlarges at an accelerated pace and the organs are withdrawn into it. If for any reason there should be a permanent disproportion in size between the viscera and the abdomen, the intestines and the liver may continue to remain out in the base of the umbilical cord.

An omphalocele has a transparent membrane through which the intestine, liver, and possibly other abdominal organs, are directly exposed to view. It may be a few centimeters in size or it may be as large as a grapefruit. The stump of the cord is attached to the apex of the sac, over which the three umbilical blood vessels course to enter the abdominal wall. Loops of small intestine are almost always extended into the sac, while portions of the stomach,

The author is Resident in Pediatrics, Abington Memorial Hospital, Abington, Pa.

spleen, pancreas and the urinary bladder may also be displaced.

Within the first twenty-four hours of life the sac wall is moist and pliable; after this time it tends to become shriveled, opaque, dried and somewhat friable. As such it is apt to rupture and be followed by evisceration and fatal peritonitis. Congenital anomalies in other parts of the body, such as clubfoot, imperforate anus, harelip or hydrocephalus may be present.

Treatment by any means other than immediate operation exposes the child to the risk of a fatal rupture of the thin sac with a spreading infection of the abdominal wall. Without operation the mortality is extremely high, and most infants succumb within a few days. The surgical repair should be performed on the first day, preferably within the first few hours. Early operation is advantageous because the stomach and intestines are not yet distended by food and gas, and hence the abdominal opening can be closed with less difficulty.

II. *Cyst of the Umbilical Cord.* This results from a large collection of Wharton's jelly which distends locally and gives rise to a cyst. It may occur within any portion of the cord, and particularly towards its attachment at the abdominal wall. It may be large enough to interfere with descent of the baby through the birth canal. If it appears at the base of the cord, there is no space between the cyst and the abdominal wall for proper placement of the ties by the obstetrician. When this occurs, the ligatures must be applied distal to the cyst and the latter must then be surgically removed from the abdominal wall. The surgical procedure should be done within the first twenty-four hours of life, since there is a tendency towards rapid necrosis and sloughing.

III. *Patent Urachus and Cyst.* In early embryonic life, the bladder normally extends up to the umbilical region, but subsequently it descends along the anterior abdominal wall. During the downward migration its upper portion becomes more and more attenuated to form a narrow, tubelike urachus which subsequently becomes obliterated. If this entire tract persists, the urinary bladder retains an opening at the navel in postnatal life. If only a segment of the tract remains, a cyst develops external to the peritoneum somewhere between the umbilicus and the superior surface of the bladder. When the entire urachus is patent, there is a constant flow of urine from the umbilical region. The diag-

nosis can be established by means of dyes which are excreted through the urine by chemical analysis of the discharge and by roentgenography following the injection of iodide solution into the bladder or the instillation of lipiodol into the umbilical opening.

Urachal cyst manifests itself as a deep, midline swelling below the navel which has a definite and broad attachment to the internal part of the abdominal wall. Urachal cysts are apt to become infected and when they do so the clinical features are those of a deep, midline abscess of the lower abdominal wall. Lateral roentgenograms can aid in making the diagnosis of a urachal cyst.

Treatment of a urachal cyst or a patent urachus is not difficult if operation is performed before infection occurs. These operations should be done in such a way that the peritoneum is not opened, for if this precaution is heeded the dangers of peritonitis are largely avoided.

IV. *Persistent Vitelline Duct.* The vitelline duct extends between the ileum and the yolk sac in the base of the umbilical cord, and remains as a sinus after the cord has been separated from the abdominal wall. In this way the intestine may attain an opening at the navel by way of a small mucosa-lined channel several inches in length. The persistent duct is occasionally the seat of inflammation but is more apt to be bothersome because of a disagreeable and malodorous discharge at the navel.

The proper diagnosis may be readily suspected by local examination and by judicious probing. It can be established without doubt if injection of lipiodol into the tract shows a communication with the intestine by fluoroscopic or film studies.

In treatment, the entire tract (including the umbilicus) must be excised; it must be detached from the ileum and a suitable closure then made of the intestinal wall.

TUMORS

Tumors of the navel of various types are recorded but are rare. The list includes angioma, enteroteratoma, dermoid cyst, myosarcoma and cysts of the urachal and omphalomesenteric remnants.

GRANULOMA

Granuloma of the umbilicus is the result of incomplete healing after separation of the cord. This is usually due to the presence

of saprophytic organisms which delays the separation of the cord and healing of the wound and this increases the liability of invasion by pathogenic organisms. It appears as a nubbin of tissue which projects from the umbilicus and interferes with epithelialization. The tissue is soft, vascular and granular, dull red or pink, and there may be an associated discharge.

Treatment consists of cauterization with silver nitrate stick repeated until the base is completely dry.

HEMORRHAGE

Hemorrhage from the navel may be due to improper ligation, trauma, weakness of the tissues, avulsion or separation of the cord. Hemophilia or hemorrhagic disease of the newborn may also be contributing causes.

Prophylaxis is of most importance; the cord must be properly ligated at time of delivery, subsequently handled gently, and asepsis of the umbilical area maintained until healing is complete. Active treatment consists of religation of the cord, cauterization or application of styptics and pressure compresses. Severe hemorrhage requires immediate transfusion of blood properly typed and cross-matched. Vitamin K should be administered parenterally. Suturing of the blood vessels may be necessary in some cases.

Hemotomas occasionally occur in the umbilical cord but require no treatment unless they are exceptionally large or interfere with proper ligation of the cord. In this latter instance the hematoma should be evacuated, otherwise it is best left alone to mummify naturally.

UMBILICAL HERNIA

Umbilical hernias, as distinguished from omphalocele or herniation into the umbilical cord, appear after a few days or weeks after the stump or the cord has dropped off. This hernia is covered with true skin and not with gelatinous tissue as in the hernia into the cord. The hernia remains as a single rounded protrusion, rarely larger than a walnut, is early reducible, and becomes prominent when the child coughs or cries. It is usually symptomless, rarely strangles, and increase in size is uncommon. The cause of the protrusion is a weakness or imperfect closure of the umbilical ring.

Various devices have been used in the non-operative treatment of umbilical hernias. Perhaps most widely used is the adhesive strapping which is placed over the umbilicus after the hernia has been reduced and each side of the adjacent abdominal wall drawn over it. The use of a coin or button placed beneath the umbilical dressing has been condemned since it prohibits adequate approximation of the margins of the hernia. Adhesive strapping should not be applied unless the umbilicus is absolutely dry and healthy, and the adhesive must be removed every week for inspection of the skin and reapplied at a different angle if there is evidence of any dermatitis. The application of tincture of benzoin to the skin affords protection against such irritation and insures against better adherence of the tape.

It is advised that if no appreciable improvement is noticed by the end of six months, surgical treatment is required which gives an excellent prognosis for permanent repair.

INFECTION

Since the advent of aseptic surgery, asepsis originating in the umbilical cord has become relatively rare. It still occurs occasionally, however, and the possibility should be borne in mind in obscure illness in infants. The pyogenic cocci and the colon bacilli have been the commonest invaders and invasion is by the umbilical arteries, vein or lymphatics. Peritonitis may arise from direct extension.

The child appears healthy for several days after birth, until it becomes evident by loss of weight, irritability, and in some cases by gastro-intestinal upset, that he is not doing well. Examination of the umbilical stump may reveal the cause; on the other hand, general sepsis can arise at this point without being evident of infection. The outlook is serious. It is apparent that prevention by careful aseptic treatment of the umbilical stump in the newborn is of utmost importance.

Treatment consists of systemic sulfonamide and/or antibiotic therapy, blood transfusions when necessary, and, if there is abscess formation, surgical incision and drainage.

CARE

It is customary to wait until the pulsation of the umbilical cord

has ceased before the clamp is applied. However, since duration of pulsation of the cord is very variable, long delay in clamping may lead to chilling of the infant. After clamping it should be firmly ligated with sterile thread or "cord ligatures" about two or three inches from the umbilicus and cut with sterile scissors an inch beyond the ligatures. Only a very small amount of blood should ooze from the cut end, and, if oozing continues, the cord must be religated. The cord stump should be wrapped in a sterile gauze sponge wrung out in alcohol or dusted with a drying powder and held in place by a flannel or gauze binder.

It is essential to keep watch on the dressings throughout the first week and especially during the first few hours since any recurrence of oozing indicates either that the ligature has become insecure or that the infant is suffering from hemorrhagic disease or sepsis. The area around the umbilicus and the proximal portion of the cord are cleansed with alcohol and then covered with a fresh dry sterile dressing. If there is even slight moistness of the umbilical stump, topical application of a bacteriostatic powder, such as sulfathiazole, is justified. The cord usually sloughs by the end of the first week of life, but a delay in separation is of no real clinical significance provided the area remains healthy.

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VOICE AND VOICE PROBLEMS IN CHILDREN*

I. W. KARLIN, M.D.
Brooklyn.

Voice development and voice disorders of children have received little attention. Perhaps this may be ascribed to the fact that voice disorders are less frequent than articulatory problems, and the therapy for voice disorders is more difficult.

The structures that are concerned with voice production are the larynx and the resonating cavities. The larynx, which houses the vocal bands, functioned originally as a valve to prevent foreign bodies from entering the respiratory tract. In the process of evolution, the larynx has assumed an additional function, that of assisting in the activities necessitating great muscular exertion. This was achieved by effecting a firm closure of the glottis. The most recent function of the larynx, biologically speaking, is voice production for speech.

In phonation, the vocal bands are approximated, leaving only a slit between them. When air is forced through the aperture, the vocal bands are set into vibration producing tone or a vocal note. These movements correspond to the pitch of the voice or the fundamental tone. The average number of movements for a male voice is about 150 and for a female voice about 300 double vibrations per second.

While the fundamental pitch of the voice is determined by the larynx alone, the characteristic tone peculiar to the speaker or singer is supplied by the resonating cavities. The vibrations of the vocal bands set up disturbances in the air column of the larynx, pharynx, nose, nasopharynx, mouth and possibly the sinuses. The shape, size and texture of these resonating cavities determine the quality of the voice. They select and modify particular overtones or partials of the fundamental or cordal tone. Certain overtones are amplified and others dampened, until there is produced the complex tone which we identify as the voice of the individual.

The first and most primitive process of phonation is the birth cry. Many interpretations have been given to the birth cry. Some claim that it is the cry of resentment at being deprived of the protective custody of the mother's womb. Its function, however, is

*From the Speech Clinic of the Pediatric Department of the Brooklyn Jewish Hospital.

physiological, having to do primarily with the establishment of normal respiration and oxygenation of the blood.

Crying consists of a deep, rapid inspiration followed by a prolonged even or interrupted expiration causing the vocal cords to vibrate. This is the first sign of an organ involved in speech producing sounds. It is found that crying occurs as early as six months in the prematurely delivered fetus, and there were reported cases of fetal crying—vagitus uterinus.

The cry, aside from being the first indication of the use of an organ concerned with speech, also has diagnostic values. One recognizes the healthy vigorous cry of the full term newborn, the low whining type of cry of the premature infant, the stridulous cry of laryngeal obstruction, the hoarse, harsh, raucous cry of the cretin, and the cephalic, high-pitched, plaintive, piercing cry which ceases as sharply as it begins and is indicative of increased intracranial pressure.

As early as the second week, the infant's crying becomes differentiated in its tonal quality. The experienced mother and the experienced nurse learn to distinguish the cry expressive of hunger, thirst, physical discomfort, pain or fatigue. Later on, crying may become purely an emotional event or a weapon for controlling the child's immediate environment.

With the onset of speech, unless there are marked abnormalities, little attention is paid to the voice of the child. Normal speaking voice has adequate loudness, clearness of tone, pitch appropriate to age and sex, slight vibrato and meaningful, graceful inflection.[1] In defining a voice disorder it is feasible to divide them into three categories:

1. Vocal defects that are due to laryngeal abnormalities, either organic or functional. Organic abnormalities include congenital anomalies of the vocal cords, defects of the glottal edges as a result of infection or nodules, and neurological or neuromuscular abnormalities interfering with the function of the vocal folds. The symptoms are predominantly hoarseness or aphonia.

The functional abnormalities include dysphonias caused by poor habits of vocalization or by emotional disturbances. The symptoms vary and have been described as breathy aspirate tone, loudness and tenseness, monotony of pitch and intensity, exaggerated changes in pitch and intensity, inappropriate pitch, inadequate loudness, intermittent vocalization and complete loss of voice.

2. Vocal defects that are due to abnormalities in the resonators. These abnormalities include rhinolalias mainly. Rhinolalia aperta is caused by cleft palate, paralysis of the velum, or inadequate neuromuscular control of the velum. Rhinolalia clausa is caused by obstruction in the nasal passages such as hypertrophied adenoid tissue, deviated septum or neoplasms. The unpleasant vocal quality in rhinolalia aperta is what is commonly termed nasality. The nasality may vary in degree all the way from slight nasalization on the vowel following or preceding a nasal sound to complete nasal emission of all sounds. Vocal symptoms in rhinolalia clausa may be divided into two classes depending on whether the nasal obstruction is in the posterior or anterior part of the nares. If the obstruction is in the posterior part, as in the case of adenoids, the nasal sounds usually become something approaching b, d and g sounds. If the obstruction is in the anterior nares, there is usually a muf-

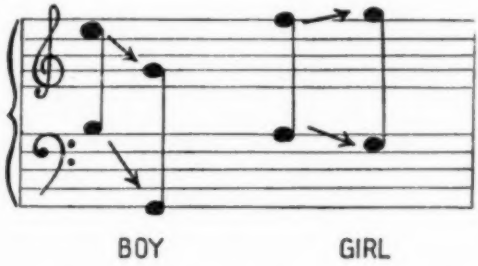

Fig. 1. Changes in singing voice at puberty (Seth and Guthrie).

fling of the nasal sounds m, n, ng, and a tendency for all the other sounds to be nasalized.

3. Another group of vocal defects is a part of a larger pathological syndrome. These defects are the typical vocal patterns perceived in the hard of hearing, the deaf, the spastic, in certain types of the feeble minded and in children with endocrinopathies. The voice of the hard of hearing and deaf is usually extremely monotonous in pitch and loudness and almost inhuman in quality. The voice of the spastic is monotonous, hence, husky, and inflectional changes are achieved with difficulty. In low grade, feeble minded children, especially in the mongoloid types, the voice is

husky. High pitched voice in the male may be an indication of enuchoidism. Hoarseness may be a symptom of hyperpituitarism or an indication of hyper- or hypothyroidism.

Under voice disorders, we may also include the changes in voice that occur during puberty. The changes are primarily physiological but added factors may be endocrine and emotional. In boys, the voice may become hoarse and raucous with a tendency to "break" into falsetto notes higher than those which were normal before the change began.

In girls there may be a break into the lower notes. However, the vocal change is usually earlier and more rapidly and more smoothly completed than in boys.[2] (Fig. 1.)

While after puberty there is a marked difference between the voices of the male and female, there is a question whether during the pre-puberty period the voices of children can be differentiated according to sex. Curry[3] states that in children the voices of the two sexes are more similar than after puberty. The opinion of trained speech clinicians is that in children before puberty, one cannot tell the sex of a child by his voice.

In the Speech Clinic of the Jewish Hospital (up to the age of 13) we ran an experiment to determine whether or not one could tell the sex of a child by his voice. A group of children, boys and girls, were placed behind a screen. The person in charge of the group assigned a number to each child. Each child was asked to recite the same line. About 20 feet away were seated 5 members of the staff of the Speech Clinic, a clerk from the office, and a nurse from the hospital—7 in all. As the number was announced, the child read the line from behind the screen and each listener wrote the number and next to it noted the sex adjudged from the voice.

There were two groups, each tested on a separate day. In the first group there were twelve children, 6 boys and 6 girls. The boys' ages were 6, 10, 11, 12, 12, 12. The girls' ages were 6, 7, 8, 9, 10, 12. Seven judged the 9-year-old girl as a male. Two judged the 7-year-old girl as a male, two judged the 10-year-old girl as a male, and two judged the 6-year-old male as a female.

In the second group, there were seven children, 3 boys and 4 girls. The boys' ages were 6, 7, 8. The girls' ages were 5, 6, 6, 7. One tester made a perfect score. Two judged one of the 6-year-old girls as a male, three judged the other 6-year-old as a male and

one judged the 7-year-old girl as a male. No male children in this group were judged as females.

A composite table of the two groups of children quoted is given below:

FEMALE

Age	5	6	7	8	9	10	12	
Number cases	1	3	2	1	1	1	1	Total 10
Number errors	0	5	3	0	7	2	0	17

MALE

Age	6	7	8	10	11	12	
Number cases	2	1	1	1	1	3	Total 9
Number errors	2	0	0	0	0	0	2

As it can be seen from the above tables, we had ten female children and seven testers. Therefore the total possible number of errors is 70. If only the laws of chance determined the choice, then there should have been 50 per cent of errors. Hence, the total probable number of errors should have been 35. Actually, there were 17 errors, or 48 per cent of the determinations were in error.

In the male group there were nine boys and seven testers. Therefore we have a total possible number of errors of 63, or a total number of probable errors of half that number, or 31.5. Actually, only two errors were obtained. Therefore, only 6 per cent of the determinations were in error in this group

It is interesting to note that the nurse and the office clerk made the same type and the same number of mistakes as the speech clinicians. In addition, from these limited experiments, several deductions suggest themselves. First, it would appear that it is possible to judge the sex of a child by his voice. Second, it is evident that where mistakes were made, it was predominantly to label a voice that belonged to a female as that of a male. Finally, it would appear that the child's voice leans toward the quality which we subjectively ascribe to be the characteristic of a male voice.

The author wishes to express his appreciation to Dr. Gladys Reid and Miss Millicent Strazzulla, for their assistance in the preparation of this paper.

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DEPARTMENT OF ABSTRACTS

NATHAN, P. W., AND RANSFORD, P. M.: SIGNIFICANCE OF CONVULSIONS IN INFANCY. (British Medical Journal, 2:421, Aug. 20, 1949).

The authors compared a group of 200 patients being treated for epilepsy as out-patients with 200 other patients who were being treated for other conditions or were supposedly well. The authors divided both groups according to age into two divisions, as follows: those between 2 to 15 years and those from 15 to 35 years. Of the control subjects 2 per cent in each age group had had infantile convulsions. Of the 100 epileptic subjects from 2 to 15, 34 had had infantile convulsions, and of the 100 from 15 to 35, 14 had had infantile convulsions. Hence, convulsions within the first two years of life are to be taken as a manifestation of epilepsy, and that the subject so afflicted is likely to have a recurrence of fits in later life. It also seems that the recurrence is likely to show itself before the fifteenth year. The authors feel that any child having convulsions should be treated with anticonvulsant drugs for a prolonged period, and possibly for years.

MICHAEL A. BRESCIA, M.D.

KAPLAN, M. Z., AND POWELEIT, A. C.: PROTEUS VULGARIS MENINGITIS. (American Journal of Diseases of Children, 77:454, April 1949).

A nine-year-old girl was admitted to the hospital with symptoms of stiff neck and headache. She gave a history of having had intermittent left otitis media for about five years and recently the left ear became tender and developed a discharge. On admission the examination revealed temperature of 104° F., papilledema, reddened right ear drum; left aural canal was so edematous that the drum was not visualized, posterior and anterior cervical nodes were enlarged and tender, and the neck was stiff and rigid. Neurologic examination revealed the presence of the Kernig and Brudzinski signs. The spinal fluid was under 550 mm. of water pressure containing 2,330 W.B.C. with 96 per cent polymorphonuclear cells. The child was presumed to have pneumococcus meningitis

and was treated with penicillin intrathecally and intramuscularly. The patient showed no improvement and developed a paralysis of the left facial and left abducens nerves. A simple left mastoidectomy was then done followed the next day by exploration of the petrous pyramid. A small cholesteatoma and two abscesses were seen along the external wall of the canal and sinus. A thrombus was found in the lateral sinus which was thought to extend to the jugular bulb. All cultures grew P. vulgaris. The child did not improve, so streptomycin was added as another medication. However, the temperature continued to spike. The thrombus of the left internal jugular vein was thought to be the cause of the septic temperature. The vein was ligated followed in two days by decided improvement. The child went on finally to recover after having had a bout of symptoms consistent with increased intracranial pressure which was thought to be due to the interference in venous return due to the thrombus and the ligation of the internal jugular vein. MICHAEL A. BRESCIA, M.D.

SHAPIRO, M. J.: DIAGNOSIS OF CONGENITAL HEART DISEASE BY ORDINARY METHODS. (Radiology, 53:469, Oct. 1949).

The author emphasizes the fact that 80 to 85 per cent of the cases of congenital heart disease can be properly diagnosed by the usual methods. The newer and more involved and sometimes dangerous procedures, such as angiocardiography and catheterization of the heart, should be reserved for the unusual and complicated cases. The lesions of congenital heart disease are divided into two main groups—those with and those without cyanosis. The lesions without cyanosis include (1) Subaortic stenosis. (2) Anomalies of the aortic arch. (3) Coarctation of the aorta. (4) Patent interventricular septum. (5) Patent interauricular septum. (6) Patent ductus arteriosus and (7) Dextrocardia with situs transversus. The lesions with cyanosis include (1) Tetrology of Fallot. (2) Tricuspid atresia with underdeveloped right ventricle. (3) Eisenmenger's complex. (4) Pulmonary stenosis. (5) Transposition of the greater vessels and (6) Truncus arteriosus. Subaortic stenosis is characterized by a loud systolic murmur and thrill over the aortic area. There is a history of heart disease from birth or early infancy but

no history of rheumatic fever. The blood pressure is normal, there is moderate left ventricular enlargement and more or less left axis deviation. Anomalies of the aortic arch are not uncommon and usually give no symptoms. Diagnosis is made by x-ray examination, and angiocardiography may help. Coarctation of the aorta is more common in males and is characterized by hypertension, decreased blood pressure in the lower extremities, moderate left ventricular enlargement, murmurs over the enlarged collateral vessels, erosion of ribs, absence of the aortic knob and left axis deviation. Interventricular septal defect reveals a loud murmur over the lower end of the sternum, normal sized heart, slight enlargement of the pulmonary artery and normal blood pressure and electrocardiogram. Interauricular septal defect is characterized by physical underdevelopment, systolic murmur over the pulmonic area, enlargement of the right heart, pulmonary artery and its branches, hypoplasia of the left ventricle and aorta, normal blood pressure and right axis deviation. Patent ductus arteriosus is more common in females and is characterized by typical machinery murmur, enlargement of the pulmonary artery and its branches with a prominent aorta, increased blood pressure, normal electrocardiogram and the heart may or may not be enlarged. Tetrology of Fallot is characterized by increasing cyanosis from birth, systolic murmur from birth, normal sized heart with relative right ventricular enlargement, diminished shadow of pulmonary artery and its branches and marked right axis deviation. Eisenmenger's complex is much less common than the tetrology of Fallot and differs from it in that there is no pulmonary stenosis. MICHAEL A. BRESCIA, M.D.

FRIEDMAN, E., and SILVERMAN, I.: THE EFFECT OF ANTIHISTAMINE MEDICATION ON THE TUBERCULIN REACTION IN CHILDREN. (American Review of Tuberculosis, *60*:354, Sept. 1949).

The authors tested the effect of an antihistaminic drug (pyribenzamine) on the tuberculin reaction in 43 children ranging in age from five and eleven years. They used old tuberculin 1:100,000. The children were first tested for positivity to the tuberculin. Several days later they were given 60 mg. pyribenzamine for two days and the tuberculin test was repeated and the drug continued at the same dosage for two more days for a total of four days. Five

controls were used in which the tuberculin test was repeated but the drug withheld. The test was repeated on ten children again using 240 mg. of pyribenzamine for a total of four days. The conclusions were that the antihistaminic did not affect the results of the tuberculin test. This result furnished further evidence that the tuberculin type reaction is not influenced by histamine. It is usually, although not always, dependent on the presence of tuberculous infection, is independent of anaphylaxis, and from the present findings is not inhibited by antihistaminic medication.

MICHAEL A. BRESCIA, M.D.

MORGAN, E. A.: PENICILLIN IN THE TREATMENT OF PRENATAL SYPHILIS. (Canadian Medical Association Journal, 61:275, Sept. 1949).

The author reports on the treatment of 58 cases of prenatal syphilis with a mortality rate of 15.5 per cent. Four cases were admitted to the hospital moribund and died before treatment could be started correcting the mortality rate to 9.0 per cent. Of 41 cases treated with penicillin or combination of penicillin and stovarsol, three died for a mortality rate of 7.0 per cent. Of twelve cases treated with stovarsol alone, 2 or 16.6 per cent died. Of 38 infants who were treated with penicillin or combination of penicillin and stovarsol, all except four became serologically negative in six weeks to eleven months, the average being five months. The dose schedule for penicillin is variable and varied from 50,000 units per pound, as total dose, to 550,000 units per pound, the average being 138,000 units per pound as the total dosage. Stovarsol was given as follows: 0.25 gm. a day for the first week, 0.25 gm. twice a day for the second week and then 0.25 gm. three times a day until the blood Wassermann became negative. The patients were treated in the hospital for two days to initiate the treatment and for study, and thereafter were treated in the out-patient department. In the child over a year of age, the penicillin was used as follows: three cases 1 to 2 million units, one case received 4 million units and 24 cases 5 to 10 million units. Penicillin effectively controls both the syphilitic infection and the intercurrent pyogenic infections, which before the days of antibiotics were the principal cause of death.

MICHAEL A. BRESCIA, M.D.

DEBRÉ, R., AND MOZZICONACCI, P.: STREPTOMYCIN TREATMENT OF SEPTICEMIA AND MENINGITIS DUE TO INTESTINAL ORGANISMS IN INFANTS. (British Medical Journal, 2:451, Aug. 27, 1949).

The authors report on 41 cases of septicemia due to gram-negative organisms treated with streptomycin and sulfonamide. Sixteen cases without meningitis were treated with 12 recoveries, whereas 25 cases of meningitis, with or without septicemia, resulted in only four recoveries. The infants ranged in age from 10 days to 15 months. The clinical picture, no matter what the organism, presents the same general features. At the onset the infection may be subacute, with failure to gain weight, or with diarrhea and vomiting that often ceases after a few days. There may be a persistent irregular fever, anorexia and progressive loss of weight. The stools may indicate faulty digestion. In some cases the infants rapidly become dehydrated and critically ill. The onset may also be marked by signs suggesting acute meningitis. However, the cases of meningitis due to the Friedländer bacillus were the only ones giving rise to clear-cut signs. Alimentary symptoms dominate the clinical picture in the septicemic forms of the disease. In several cases edema of the abdominal wall was present, particularly in the subumbilical region, and sometimes edema of the genitalia as well. Other findings included purpura of the abdominal wall, hepatomegaly, splenomegaly, jaundice and areas of localized gangrene. The signs of these infections are often obscure at the onset, and many of these cases can be discovered only by careful examination of infants suffering from toxemia and subacute fever with alimentary disturbances.

MICHAEL A. BRESCIA, M.D.

Archives of Pediatrics

A Monthly Journal Devoted to the

Diseases of Infants and Children

EDITED BY

JOHN FITCH LANDON, A.B., M.D.

Attending Pediatrist, Roosevelt Hospital and Woman's Hospital; Visiting Physician, Willard Parker Hospital; Fellow New York Academy of Medicine, New York City.

VOLUME LXVI

JANUARY TO DECEMBER

1949

E B TREAT & CO., INC., Publishers

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NEW YORK

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ARNOLD, JESSE H.

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INDEX TO VOLUME LXVI

Albanese, John P. Amyotonia congenita. Report of two cases..... 411
Amigen-dextri-maltose feeding of the newborn. Dunbar and Lynxwiler 212
Amyotonia congenita. Report of two cases. Albanese..... 411
Anemia, sickle cell, the clinical manifestations of..... 89
Antihistamine medication, the effect of, on the tuberculin reaction in children 574
Anus and rectum, congenital anomalies of the. A Study of seventeen cases and comparative review of the literature. Vasilas..... 476
Apfel, Harry. A simplified method of the care of the skin of the newborn 131
Apfel, Harry. Influenzal meningitis. Evaluation of its treatment..... 392
Arnold, Jesse H. and Grotts, Bruce F. Functional hypoglycemia in children 462
Asthma, infantile, apparent cure of, with the use of a preparation derived from pregnant woman's urine..... 532
Astroblastoma of the cerebellum in an infant. Report of case. Grotts 283
Atelectasis and hypoglycemia, obstruction of the bile ducts. Litchfield and Moss..... 97
Bacillus dysenteriae (Shiga) in infants, a clinical study of sixty-two cases of intestinal infection by the. LaFétra and Howland..... 512
Bacillus of Shiga, relation of the, to the summer diarrheas of children. Flexner, Simon; Park, W. H.; Koplik, Henry; Holt, L. Emmett; Knox, J. H. M. and Booker, W. H..... 363
Baxter, E. H. and Walliker, George W. Methemoglobinemia. A cause of cyanosis in infants and children..... 143
BCG vaccination 134
Beirne, Donald P.; Brunhofer, Andrew C.; Schwartzman, Joseph and Crusius, Margaret E. Friedlander's bacillus meningitis in newborns 9
Benson, Reuel A.; Schneider, Mae and Brand, David. The use of rice as a cereal food in infants..... 209
Bile ducts, obstruction of the, hypoglycemia and atelectasis. Litchfield and Moss..... 97
Blalock operation for congenital cyanosis..... 545
Bloch, Harry. Celiac syndrome due to gastro-intestinal allergy..... 54
Boelter, Muriel and Hatoff, Alexander. The Schwartzman phenomenon with penicillin..... 335
Blood findings in juvenile amaurotic idiocy..... 348
Book reviews 95, 142, 279, 386, 436, 488, 536
Booker; Knox, J. H. M.; Flexner, Simon; Park, W. H.; Koplik, Simon; and Holt, L. Emmett. Relation of the bacillus of Shiga to the summer diarrheas of children..... 363
Bow legs, physiologic. Forrest..... 349
Brand, David; Schneider, Mae and Benson, Reuel E. The use of rice as a cereal food in infants..... 209
Brescia, Michael A. and Tartaglione, Edward F. Functional intestinal obstruction in an infant..... 387

Brunhofer, Andrew C.; Schwartzman, Joseph; Crusius, Margaret E. and Beirne, Donald P. Friedlander's bacillus meningitis in newborns 9
Budin's method of infant feeding. Zahorsky 57
Celiac disease, folic acid in 488
Celiac syndrome due to gastro-intestinal allergy. Bloch 54
Chickenpox and herpes zoster, relation between 172
Choline in toxicosis of infants 208
Chorea, Sydenham's, two factors in the differentiation of rheumatic and non-rheumatic types of. Sherman and Kaiser 173
Cioffari, Mario S. Dietary control of dental caries 233
Clindactyly, hereditary 281
Clinical-pathological conference, Willard Parker Hospital 44, 82, 184, 270, 332, 380, 484, 526
Clubbing of the fingers. Thompson 216
Coccidioidomycosis. Case studies in children. Cohen 241
Cohen, Robert. Coccidioidomycosis. Case studies in children 241
Convulsions in infancy, significance of 572
Convulsions, febrile, transient hemiplegia associated with. Report of case. Schwartzman 489
Corpulence in childhood and adolescence 385
Crusius, Margaret E.; Beirne, Donald P.; Brunhofer, Andrew S. and Schwartzman, Joseph. Friedlander's bacillus meningitis in newborns 9
Curare treatment of poliomyelitis 141
Cyanosis, congenital, Blalock operation for 545
Davolos, Dominick. The epidemiology of poliomyelitis 30, 70
Dental caries, dietary control of. Cioffari 233
Dermatitis, napkin, caused by ammoniacal urine. Treatment with methionine. Goldstein 553
Diabetes, juvenile, lipocaic insufficiency in 401
Diarrhea, acute, the treatment of, in infancy. Hanafi 352
Diarrhea, penicillin by mouth for infants with 265
Diarrheas, summer, of children, relation of the bacillus of Shiga ot the. Flexner, Simon; Park, W. H.; Koplik, Henry; Holt, L. Emmett; Knox, J. H. M. and Booker, W. H. 363
Dietary control of dental caries. Cioffari 233
Diphtheria, faucial, in adults with reference to the early diagnosis 93
Diphtheria in infants 53
Dunbar, Ernest A. and Lynxwiler, Chester P. Amigen-dextri-maltose feeding of the newborn 212
Dunbar, Ernest A., Jr. and Lynxwiler, Chester P. Plasma-olac feedings of the premature 20
Dyspepsia, infantile, a new sulfonamide in the treatment of. El-Gholmi, Hanna, El-Kholy and El-Sobky 492
Dysplasias, early, of the hip. Koven and Koven 201
Dysplexia, causes and symptoms. Park 289
El-Gholmi, Abdel Azim; Hanna, Maurice; El-Kholy, Salah El Dine and El-Sobky, Zeinab. A new sulfonamide in the treatment of infantile dyspepsia 492

El-Kholy, Salah El Dine; El-Sobky, Zeinab; El-Gholmi, Abdel Azim and Hanna, Maurice. A new sulfonamide in the treatment of infantile dyspepsia 492
El-Sobky, Zeinab; El-Gholmi, Abdel Azim; Hanna, Maurice and El-Kholy, Salah El Dine. A new sulfonamide in the treatment of infantile dyspepsia 492
Endocarditis, subacute bacterial, superimposed on Roger's disease treated with penicillin and sumédine 533
English, Dorothy A. Tuberous sclerosis 24
Enuresis, a new cure for 278
Epilepsy in children, electroencephalography in diagnosis of 19
Epileptic children, emotional problems in treatment and prognosis of. Symonds 355
Erythema nodosum after administration of sulfathiazole 392
Erythroblastosis in the newborn, simple method of detection 240
Eye lesions in acute leukemia of infants 535
Flexner, Simon; Park, W. H.; Koplik, Henry; Holt, L. Emmett; Knox, J. H. M. and Booker. Relation of the bacillus of Shiga to the summer diarrheas of children 363
Folic acid in celiac disease 488
Forrest, Herbert J. Physiologic bow legs 349
Freedman, Myron and Laderman, Peter. Thrombocytopenic purpura complicating rubella 135
Friedlander's bacillus meningitis in newborns. Report of case in one of colored premature twins and review of the literature. Schwartzman, Crusius, Beirne and Brunhofer 9
Gastro-intestinal allergy, celiac syndrome due to. Bloch 54
Gillespie, James B. and Sargent, Frederick K. Recurrent paroxysmal tachycardia in infancy 437
Goldstein, Louis S. Napkin dermatitis caused by ammoniacal urine. Treatment with methionine 553
Gonorrhea in children, penicillin in 61
Grotts, Bruce F. Astroblastoma of the cerebellum in an infant 283
Grotts, Bruce F. Fetal peritonitis. Report of four cases 1
Grotts, Bruce F. and Arnold, Jesse H. Functional hypoglycemia in children 462
Hamill, Samuel McC. and Nicholson, William R. Infections of the newborn 419
Hanafi, Mamdouh. The treatment of acute diarrhea in infancy 352
Hanna, Maurice; El-Kholy, Salah El Dine; El-Sobky, Zeinab and El-Gholmi, Abdel Azim. A new sulfonamide in the treatment of infantile dyspepsia 492
Hatoff, Alexander and Boelter, Muriel. The Schwartzman phenomenon with penicillin 335
Heart disease, congenital, diagnosis of, by ordinary methods 573
Heart disease, congenital, physiological studies in 90
Heart disease, congenital. Mautner 546
Hemiplegia, transient, associated with febrile convulsions. Report of case. Schwartzman 489

Hemorrhage, adrenal, in monocytic leukemia..... 385
Hepatic and skeletal defects, connection between..... 23
Herpes zoster and chickenpox, relation between..... 172
Holt, L. Emmett; Knox, J. H. M.; Flexner, Simon; Park, W. H.; Koplik, Simon and Booker. Relation of the bacillus of Shiga to the summer diarrheas of children..... 363
Howland, John and LaFétra, L. E. A clinical study of sixty-two cases of infantile infection by the bacillus dysenteriae (Shiga) in infants 512
Hyperinsulinism, spontaneous (Harris' syndrome)..... 91
Hyperthyroid, transitory, syndrome in an infant of one and a half months 534
Hypoglycemia and atelectasis, obstruction of the bile ducts. Litchfield and Moss 97
Hypoglycemia, functional, in children. Report of two cases. Grotts and Arnold 462
Hypothyroidism, mild, in children..... 92
Idiocy, amaurotic, blood findings in juvenile..... 348
Infant care, opinions of pediatricians on certain problems of. Weinfeld 266
Infantile paralysis, treatment of..... 130
Infections and injuries of newborn children. Snow..... 318
Infections of the newborn. Hamill and Nicholson..... 419
Influenzal meningitis. Evaluation of its treatment. Apfel..... 393
Injuries and infections of newborn children. Snow..... 318
Interventricular septal defect with Stokes-Adams syndrome. Siegel.... 62
Intestinal infection by the bacillus dysenteriae (Shiga) in infants, a clinical study of sixty-two cases of. LaFétra and Howland..... 512
Intestinal obstruction, functional, in an infant. Brescia and Tartaglione. 387
Kaiser, I. Howard and Sherman, Robert L. Two factors in the differentiation of rheumatic and non-rheumatic types of Sydenham's chorea. 173
Karlin, I. W. Voice and voice problems in children..... 567
Knox, J. H. M.; Flexner, Simon; Park, W. H.; Koplik, Simon; Holt, L. Emmett and Booker. Relation of the bacillus of Shiga to the summer diarrheas of children..... 363
Koplik, Henry; Holt, L. Emmett; Knox, J. H. M.; Flexner, Simon; Park, W. H. and Booker. Relation of the bacillus of Shiga to the summer diarrheas of children..... 363
Koven, Benjamin and Koven, Morris T. Early dysplasias of the hip.. 201
Koven, Morris T. and Koven, Benjamin. Early dysplasias of the hip.. 201
Laderman, Peter and Freedman, Myron. Thrombocytopenic purpura complicating rubella. 135
LaFétra, L. E. and Howland, John. A clinical study of sixty-two cases of intestinal infection by the bacillus dysenteriae (Shiga) in infants. 512
Laurence-Moon-Bardet-Biedl syndrome 109
Lenarsky, Maurice. Rocking bed—its use in poliomyelitis..... 339
Leprosy treated with penicillin..... 92
Leukemia, acute, of infants, eye lesions in..... 535
Leukemia and salmonellosis (S. Dublin)..... 134
Leukemia, monocytic, adrenal hemorrhage in..... 385

Leukotomy in children 418
Lipocaic insufficiency in juvenile diabetes 401
Litchfield, Harry R. and Moss, Armand H. Obstruction of the bile ducts, hypoglycemia and atelectasis 97
Lymphocytosis, acute infectious 89
Lynxwiler, Chester P. and Dunbar, Ernest A. Amigen-dextri-maltose feeding of the newborn 212
Lynxwiler, Chester P. and Dunbar, Ernest A., Jr. Plasma-olac feedings of the premature 20
Malformations, congenital, after exposure to measles in early pregnancy. 475
Malformations, congenital, in infant, rubella in mothers as cause of 282
Malformation, congenital, pathogenesis of 282
Mautner, Hans. Congenital heart disease 546
Measles, congenital malformations after exposure to, in early pregnancy. 475
Meningitis and septicemia, streptomycin treatment of, due to intestinal organisms in infants 576
Meningitis, serous, in epidemic pleurodynia 552
Meningitis, B. Coli, treatment and cure of, with streptomycin 532
Meningitis, proteus vulgaris 572
Meningitis, influenzal. Evaluation of its treatment. Apfel 392
Meningitis, tuberculous, psychic disturbances associated with 172
Meningitis, tuberculous, results of treatment of, in infants and children with streptomycin 277
Meningitis, tuberculous, streptomycin in 94, 141
Meningitis, tuberculous, the treatment of, with streptomycin 276
Methemoglobinemia. A cause of cyanosis in infants and children. Walliker and Baxter 143
Methionine, napkin dermatitis caused by ammoniacal urine. Treatment with. Goldstein 553
Micetina, the treatment of salmonella infections with 534
Milk embolism as cause of sudden death in infants 560
Mongol delivered of a mongolian infant 535
Monster, two-headed 276
Moss, Armand H. and Litchfield, Harry R. Obstruction of the bile ducts, hypoglycemia and atelectasis 97
Muscle spasm, disorders of respiration in bulbar and other forms of poliomyelitis, with special reference to. Pohl 537
Nau, Cornelius H. Poliomyelitis following tonsillectomy 49
Neuritis, multiple, and poliomyelitis. Scobey 110, 157
Neurobartonellosis 483
Neuroblastomas 487
Nicholson, William R. and Hamill, Samuel McC. Infections of the newborn 419
Osteomyelitis, acute, of infants and adolescents 362
Osteomyelitis, hematogenous, penicillin in 69
Paralysis, infantile, in Mauritius 91
Park, George E. Causes and symptoms of dysplexia 289
Park, George E. Perplexing factors in reading retardation 500

Park, W. H.; Koplik, Henry; Holt, L. Emmett; Knox, J. H. M.; Flexner, Simon and Booker. Relation of the bacillus of Shiga to the summer diarrheas of children 363
Pascucci, Stephen E. Anomalies and diseases of the umbilical cord 561
Pediatrics, penicillin aerosols in 566
Pellagra, gastric mucous membrane in treatment of 525
Penicillin aerosols in diseases of lung 499
Penicillin aerosols in pediatrics 566
Penicillin and sumédine, subacute bacterial endocarditis superimposed on Roger's disease treated with 533
Penicillin in the treatment of prenatal syphilis 575
Penicillin by mouth for infants with diarrhea 265
Penicillin in gonorrhea in children 61
Penicillin in hematogenous osteomyelitis 69
Penicillin in scarlet fever 351
Penicillin poisoning in a case of acute staphylococcus aureus hemolyticus infection of a hip joint 93
Penicillin, Schwartzman phenomenon with. Report of case. Boelter and Hatoff 335
Peritonitis, fetal. Report of four cases. Grotts 1
Plasma-olac feedings of the premature. Report of two cases. Dunbar and Lynxwiler 20
Pleurisy, tuberculous, local streptomycin therapy in 215
Pleurodynia, epidemic, serous meningitis in 552
Pneumonia in the newborn from inhalation of gastric contents 435, 491
Pohl, John F. Disorders of respiration in bulbar and other forms of poliomyelitis, with special reference to muscle spasm 537
Poliomyelitis 156
Poliomyelitis, acute anterior and acute posterior. Are they the same disease? Scobey 402
Poliomyelitis and multiple neuritis. Scobey 110, 157
Poliomyelitis, anterior, treatment of. Report on intraspinal administration of pyridoxine and thiamine hydrochloride and artificial fever therapy. Stone 443
Poliomyelitis, curare treatment of 141
Poliomyelitis, disorders of respiration in bulbar and other forms of, with special reference to muscle spasm. Pohl 537
Poliomyelitis following tonsillectomy. Nau 49
Penicillin, leprosy treated with 92
Poliomyelitis morbidity, increased, in older age groups 511
Poliomyelitis, respiratory disturbances in 211
Poliomyelitis, rocking bed—its use in. Lenarsky 339
Poliomyelitis, second attacks of 48
Poliomyelitis, the epidemiology of. Davolos 30, 70
Porencephaly, congenital, in children 188
Purpura, thrombocytopenic, complicating rubella. Freedman and Laderman 135
Radioactive iron, absorption of, by children 276

Rectum and anus, congenital anomalies of the. A study of seventeen cases and comparative review of the literature. Vasilas..... 476
Reading retardation, perplexing factors in. Park..... 500
Respiratory disturbances in poliomyelitis..... 211
Rh factor blood incompatibility..... 88
Rheumatic fever, etiology of. Present day concepts. Wagner..... 301
Rheumatism in childhood..... 269
Rice, the use of, as a cereal food in infants. Benson, Schneider and Brand 209
Rocking bed—its use in poliomyelitis. Lenarsky..... 339
Roger's disease, subacute bacterial endocarditis superimposed on, treated with penicillin and sumédine..... 533
Rubella during pregnancy and congenital defects in child..... 188
Rubella in mother as cause of congenital malformations in infant..... 282
Rubella, thrombocytopenic purpura complicating. Freedman and Laderman 135
Salmonella infections, the treatment of, with micetina..... 534
Salmonellosis (S. Dublin) and leukemia..... 134
Sargent, Frederick K. and Gillespie, James B. Recurrent paroxysmal tachycardia in infancy..... 437
Scarlet fever, penicillin in..... 351
Schneider, Mae; Brand, David and Benson, Reuel E. The use of rice as a cereal food in infants..... 209
Schwartzman, Joseph; Crusius, Margaret E.; Beirne, Donald P. and Brunhofer, Andrew C. Friedlander's bacillus meningitis in newborns. 9
Schwartzman, Joseph. Transient hemiplegia associated with febrile convulsions 489
Schwartzman phenomenon with penicillin, Report of case. Boelter and Hatoff 335
Sclerosis, tuberous. Report of case. English..... 24
Scobey, Ralph R. Acute anterior and acute posterior poliomyelitis. Are they the same disease?..... 402
Scobey, Ralph R. Multiple neuritis and poliomyelitis..... 110, 157
Septicemia and meningitis, streptomycin treatment of, due to intestinal organisms in infants..... 576
Sherman, Robert L. and Kaiser, I. Howard. Two factors in the differentiation of rheumatic and non-rheumatic types of Sydenham's chorea. 173
Siegel, Earl. Interventricular septal defect with Stokes-Adams Syndrome 62
Skin of the newborn, the care of, a simplified method. Apfel..... 131
Smallpox epidemics, forecasting, on climatic data..... 109
Snow, Irving M. Injuries and infections of newborn children..... 318
Stokes-Adams syndrome, interventricular septal defect with. Siegel..... 62
Stone, Simon. The therapeutic and prophylactic administration of wheat germ oil (vitamin E) in infancy and childhood..... 189
Stone, Simon. Treatment of anterior poliomyelitis. Report on intraspinal administration of pyridoxine and thiamine hydrochloride and artificial fever therapy..... 443

Streptomycin in abdominal tubersulosis..... 183
Streptomycin in pulmonary tuberculosis in children..... 232
Streptomycin in tuberculosis of bone..... 533
Streptomycin in tuberculous meningitis.....94, 141
Streptomycin, results of treatment of tuberculous meningitis in infants and children with..... 277
Streptomycin, the treatment of tuberculous meningitis with..... 276
Streptomycin treatment of septicemia and meningitis due to intestinal organisms in infants..... 576
Streptomycin therapy, local, in tuberculous pleurisy..... 215
Streptomycin, treatment and cure of a case of B. coli meningitis with streptomycin 532
Sulfathiazole, erythema nodosum after administration of..... 392
Sulfonamide, new, in the treatment of infantile dyspepsia. El-Gholmi, Hanna, El-Kholy and El-Sobky..... 492
Sunnédine and penicillin, subacute bacterial endocarditis superimposed on Roger's disease treated with..... 533
Symonds, Alexandra. Emotional problems in treatment and diagnosis of epileptic children 355
Syphilis, prenatal, penicillin in the treatment of..... 575
Tachycardia, recurrent paroxysmal, in infancy. Sargent and Gillespie.. 437
Tartaglione, Edward F. and Brescia, Michael A. Functional intestinal construction in an infant..... 387
Thompson, Ian. Clubbing of the fingers..... 216
Tonsillectomy, poliomyelitis following. Nau..... 49
Toxicosis of infants, choline in..... 208
Tuberculin reaction in children, the effect of antihistamine medication on the..... 574
Tuberculosis, abdominal, streptomycin in..... 183
Tuberculosis, human, due to avian bacilli..... 531
Tuberculosis of bone, streptomycin in..... 533
Tuberculosis, pulmonary, in children, streptomycin in..... 232
Umbilical cord, anomalies and diseases of the. Pascucci..... 561
Vasilas, Anthony. Congenital anomalies of the rectum and anus. A study of seventeen cases and comparative review of the literature... 475
Virus diseases and their effect on fetus..... 183
Voice and voice problems in children. Karlin..... 567
Wagner, Bernard M. Etiology of rheumatic fever. Present day concepts. 301
Walliker, George W. and Baxter, E. H. Methemoglobinemia. A cause of cyanosis in infants and children..... 143
Weinfeld, Gustave F. Opinions of pediatricians on certain problems of infant care 266
Wheat germ oil (vitamin E), the therapeutic and prophylactic administration of, in infancy and childhood. Stone..... 189
Zahorsky, John. Budin's method of infant feeding..... 57

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